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EXPERIMENTAL MENINGITIS IN GUINEA PIGS[1]

By S. E. BRANHAM, *Senior Bacteriologist*, R. D. LILLIE, *Surgeon*, and A. M. PABST, *Junior Bacteriologist*,[2] *United States Public Health Service, National Institute of Health*

In November 1931 a study of the effects of intra cisterna magna injections of meningococci and meningococcus products in rabbits and guinea pigs was begun. The studies with rabbits, published in 1932 (*1*, *2*), showed that a meningitis could be induced by living or heat-killed meningococci and also by filtrates of meningococci. Our earlier experiments with guinea pigs were reported briefly in 1932 (*3*). The present paper represents a continuation of these studies.

EXPERIMENTAL WORK WITH GUINEA PIGS

(*A*) *Animals, material, and technique used.*—Young guinea pigs, weighing 200 to 250 grams, were injected intracisternally, under ether anesthesia, with living cultures of meningococci, with heat-killed cultures, with filtered suspensions of living cultures, and with broth filtrates. Weight and temperature were recorded daily for each animal as long as it was under observation.

The cultures were of freshly isolated strains. For injection, these cultures were grown for 18 hours on blood agar slants, and suspensions in Ringer's solution were prepared as for rabbits (*2*). The dose given to guinea pigs usually varied from 10,000,000 to about 100,000,000 meningococci, depending on the virulence of the strain. The number of meningococci injected was contained in a volume of 0.2 to 0.3 cc of Ringer's solution. Larger volumes by this route were avoided as far as possible in order to prevent the occurrence of increased intracranial pressure.

The killed cultures were prepared in the same way as were the living, but the suspensions were boiled for 5 minutes before being injected. The volume of the dose was the same as that with the living bacteria, but the number of meningococci contained in it was usually much greater—sometimes several billion.

The filtered suspensions were prepared as for the production of the Shwartzman reaction (*4*), but no preservative was added and suspensions were filtered through Berkefeld N filters. A very small amount of these filtered suspensions represented the washings of a tremendous

[1] Read before the Society of American Bacteriologists, at Ann Arbor, Mich., December 1932.

[2] Miss Pabst died on Dec. 25, 1935, of meningococcus septicemia, acquired in line of duty.

number of bacteria. The broth filtrates were prepared by the method described by Ferry (*5*).

The amount of filtered suspensions and of filtered broth cultures used for injection did not exceed 0.3 cc in any case, and was usually 0.1 to 0.15 cc.

(*B*) *Effects of injections with living cultures.*—The newly isolated strains of meningococci varied greatly in their virulence for guinea pigs. This was true even of the primary cultures from spinal fluids. Certain strains—i. e., strains 403 and 541—in amounts of 10,000,000 meningococci regularly killed 100 percent of the animals. Other strains in similar doses killed only a small percentage, while still others failed to induce any symptoms.

The clinical pictures found in guinea pigs were less constant than those noted in rabbits. The "incubation period" after injection was usually 3 to 5 hours. During this time the animals seemed quite well, though not always lively. The first signs noted were dyspnea and a tendency for the head to be held to one side. This stage, which was usually transient, was not accompanied by rigidity of the neck. The animal's fur was ruffled and it sat huddled in obvious discomfort. That this discomfort was chiefly in the back of the head was indicated by the character of the efforts made by the animals to get rid of it, for they dodged from side to side and frequently tried to scrape it off with their paws. In some guinea pigs there were no other symptoms, and the animals seemed quite recovered by the next day. In some, this stage was followed by violent shivering and hypersensitiveness to touch; in others, peculiar convulsive, rhythmical jerking appeared. As the clinical picture developed, the jerking was often followed by prostration, and by a more or less complete paralysis of the hind legs. Coughing, convulsions, and hemorrhage from the nose often appeared. Although death occurred as early as 4 hours after injection and as late as 4 days, as a rule it came most often between 8 and 24 hours. Recovery took place in some animals that had extensive paralysis, whereas death occurred in some whose symptoms were much less marked. Generally, the prognosis was poor for those guinea pigs in which paralysis had developed.

In some of these animals the disease ran a more chronic course. These developed marked opisthotonos and rigidity, and went into tetanic spasms when touched or jolted. Usually the temperature fell several degrees. One of these guinea pigs (H–15) is described in protocol 3. Protocols 1 (D–3) and 2 (J–1) present guinea pigs showing the more acute pictures. In general, it may be said that the injection of living virulent cultures of meningococci into guinea pigs may result in (*a*) a convulsive form (protocol D–3), (*b*) a paralytic form (protocol J–1), and (*c*) a more chronic form with spasticity, rigidity, and opisthotonos (protocol H–15).

(*C*) *Effects of injections with killed cultures.*—Twenty guinea pigs were given intracisternal injections of suspensions of the strains used in (B) boiled 5 minutes. Eight of these developed symptoms resembling the paralytic form of meningitis described above, and seven of them died within 12 hours. The animals that died received at least 200,000,000 meningococci in a volume of 0.2 to 0.3 cc of Ringer's solution. Those receiving a smaller number of the bacteria remained well or were only slightly affected. It is well known that the thermostable "endotoxins" of meningococci will kill animals when given in sufficiently large quantities by various routes. This was noted as early as 1901 by Albrecht and Ghon (*6*), and has been discussed by many workers (*7*) since, e. g., Kolle and Wassermann, Flexner, Kraus and Doerr, Dopter, Gordon, Neill and Taft, and others.

(*D*) *Effects of injections with filtered suspensions.*—In order to get a clearer idea of whether the "toxins" of the meningococcus remained chiefly within the intact bacterial cells, or were dissolved in the menstruum of the suspension, Berkefeld N filtrates of suspensions of 4 different strains were given intracisternally to 24 guinea pigs. These filtrates represented massive doses, as the suspensions from which they were made were heavy. Only 4 of these guinea pigs remained unaffected, and of the 20 which developed definite symptoms, 18 died within 14 hours after injection. The outstanding symptoms were marked dyspnea and extreme prostration after an incubation period which was usually 3 to 4 hours.

(*E*) *Effects of injections of broth culture filtrates.*—It was at this time that Dr. Ferry and his associates reported the production of soluble toxins in broth cultures by meningococci. Berkefeld N filtrates of 6- to 10-day broth cultures were prepared by us after their method (*5*), choosing for study those from strains which produced the heaviest pellicles. From 0.1 to 0.3 cc of these, and of various dilutions of them, were given to guinea pigs intracisternally as described above.

These filtrates varied widely in their toxicity. Some of the "toxins" were too weak to do more than produce the mildest symptoms when given undiluted; others killed as many as 80 percent of the guinea pigs that were given 0.1 to 0.2 cc of 1:5 dilutions. More than 300 guinea pigs were studied altogether with these "toxins." The clinical picture found in them was essentially that found in the animals given the materials described above—in other words, that of a typical meningitis. The incubation period varied from 2½ to 6 hours, and death usually occurred in 8 to 24 hours.

(*F*) *Effect of injection of control materials.*—Ten guinea pigs were injected intracisternally as follows: 3 with diluted india ink, 0.1 cc, 0.2 cc, and 0.3 cc, respectively; one with 0.1 cc of undiluted ink; 3 with 0.85 percent NaCl solution, 0.3 cc; and 3 with 0.1 cc, 0.2 cc, and 0.3 cc, respectively, of 1 percent glucose broth. None of the animals

showed any symptoms whatever. Obviously neither the volume given nor the mere presence of inert foreign material played a role in producing meningitis in guinea pigs as described above.

(*G*) *Autopsy findings.*—Most of the animals that died were autopsied, and some that showed no symptoms, or that recovered, were chloroformed and studied similarly. Cultures on rabbits' blood agar were made from cisternal fluid drawn just before autopsy from the surface of the brain and from the heart. Smears of cisternal fluid or from the surface of the brain were stained by Wright's and Gram's methods. In those instances in which postmortem changes could be ruled out, the brain was removed for histological examination.

Gross findings at autopsy were not conspicuous and consisted chiefly of adherence of the meninges, sometimes pus, especially between the cerebellum and the hemispheres, and usually congestion.

The stained smears usually showed large numbers of leucocytes; but, unless the animal was autopsied immediately after death, these leucocytes had degenerated so far that the presence or absence of bacteria could not always be thus detected. Gram-negative diplococci of characteristic morphology were seen in smears from 65 percent of the guinea pigs which died after receiving living cultures. Occasionally a number of lymphocytes were also present.

(*H*) *Bacteriological studies.*—Meningococci were recovered from the cisternal fluid drawn just before autopsy from about 25 percent of the guinea pigs dying after injection with living cultures. In about 5 percent they were found in the blood. Most of these strains were found by agglutination to be identical in serological grouping with the strains that were injected. Other strains were nonagglutinable. All were recovered from pigs that died within 24 hours after injection.

Microorganisms other than the meningococcus were recovered from both cisternal fluid and from blood in a number of guinea pigs. These were usually strains belonging to the *Pasteurella* group, though there was occasionally a hemolytic streptococcus or a staphylococcus. These occurred regardless of the nature of the inoculum, and practically always in animals that died after several days when recovery from the primary meningitis seemed well under way. Death in these cases was seemingly due to secondary or superimposed infection. On account of such cases it seemed essential that a careful bacteriological study be made of all guinea pigs that died in the course of these experiments before assuming that their death was due to meningococci or their products.

PATHOLOGIC HISTOLOGY

In guinea pigs, as in rabbits, a purulent meningitis was produced alike by living meningococci, by killed cultures, by Berkefeld filtrates of suspensions from agar cultures (hereafter referred to as suspension filtrates), and by filtrates of broth cultures.

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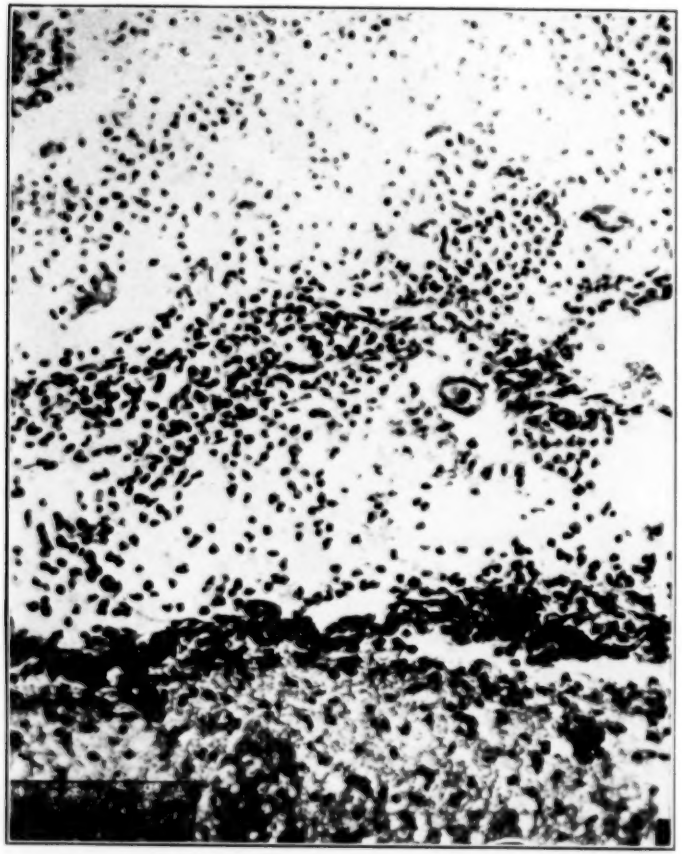

FIGURE 1.—Guinea pig A1. Fibrinopurulent meningitis, base of midbrain. Died 11–12 hours after injection.

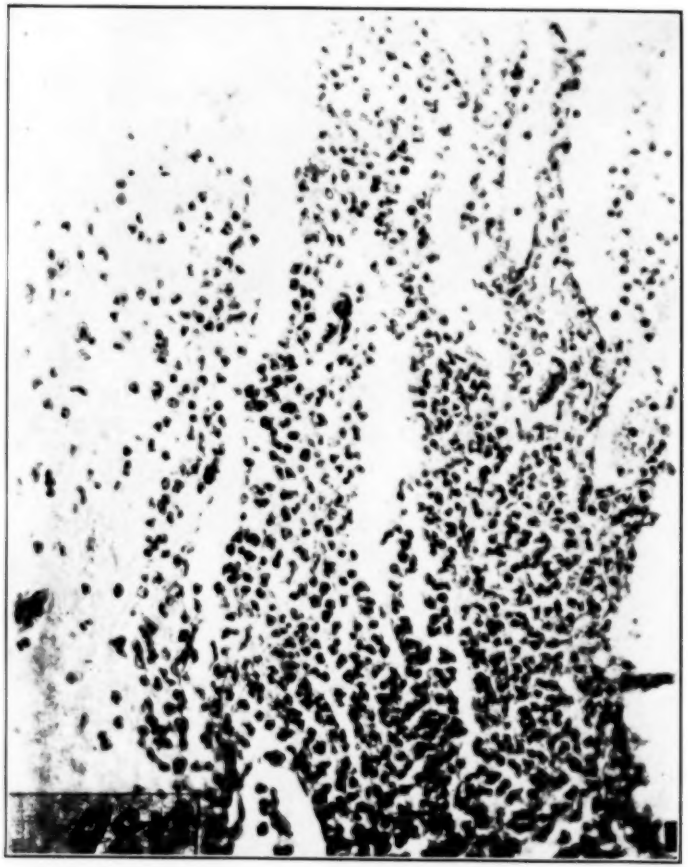

FIGURE 2.—Guinea pig A7. Purulent meningeal infiltration, midbrain. Died 48 hours after injection.

FIGURE 3.—Guinea pig A7. Purulent infiltration of choroid plexus, with purulent exudate in lateral ventricle.

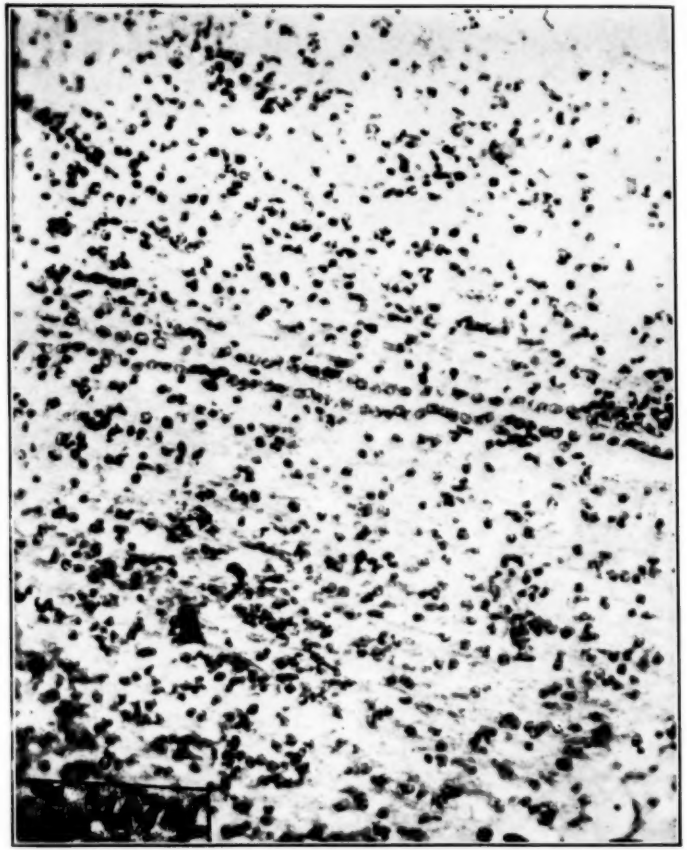

FIGURE 4.—Guinea pig A6. Purulent infiltration of wall of lateral ventricle.

The meningeal exudate appeared in 3 to 5 hours, much earlier than in rabbits. It was predominantly purulent in character (figs. 1 and 2), denser on the base of the brain and between the brain stem and the occipital cortex, and was often accompanied by congestion and hemorrhage. Fibrin was most often evident after inoculation with killed cocci. Demonstrable fibrin exudation was infrequent with the other inocula. Similarly, purulent thrombi in meningeal vessels were frequent after inoculation with killed cocci and infrequent with the other inocula.

The sheaths of perforating and, less often, deep vessels were often infiltrated by purulent exudate. Occasionally the purulent infiltration extended to the perivascular parenchyma. Purulent thrombosis was not infrequent, being especially common after inoculation with killed cocci.

Purulent exudate in the ventricles (fig. 3) occurred in the great majority of the animals inoculated with living or killed cultures and in about 75 percent of those receiving broth filtrates and dying in less than 30 hours, but was infrequent with the suspension filtrates. Choroid plexus infiltration was correspondingly frequent. With the broth filtrates this infiltration was as often predominantly purulent as lymphocytic in character, while with living or killed cocci lymphocytes tended to dominate.

Purulent infiltration of the brain substance abutting on the meninges or ventricles (fig. 4) was found in over 50 percent of the animals inoculated with cultures or broth filtrates. The few inoculated with suspension filtrates showed this condition infrequently. Such marginal purulent infiltration was more frequent and more marked in guinea pigs than in rabbits.

Other less frequent features of the process were perivascular hemorrhages, miliary marginal or central abscesses, and suppurating ependymal ulcers in the ventricles.

Pericellular edema in the cerebral cortex and tigrolysis and vacuolation in the nuclei of the brain stem were frequent findings, especially in animals surviving over 12 hours.

In subsiding reactions the meningeal exudate decreased in amount and became partly or entirely lymphocytic in character, the involvement of intracerebral vessel sheaths disappeared and the ventricular exudates decreased and disappeared. The ventricles were often reduced to slit-like spaces, and the plexal villi sometimes agglutinated into masses with some pus between. Plexal infiltration became lymphocytic or disappeared entirely. Purulent infiltrations of the brain substance were infrequent in late stages. With filtrates and killed cultures evidence of subsidence was noted early on the second day, while with living cultures no decrease in the reaction was apparent until after 3 days. In 8 of the 9 extensive purulent meningitides persisting to

the third to fifth day after broth filtrate inoculation, secondarily invading organisms were demonstrated in the meninges either by culture or in sections.

This account is based on the study of the brains from 44 guinea pigs inoculated with living cultures, 12 with heat-killed cultures, 18 with suspension filtrates, and 104 with broth filtrates.

DISCUSSION

Meningitis can be produced in guinea pigs more readily than in rabbits by intracisternal injection of meningococci or their products.

Apparently a true infection results if the strains used are sufficiently virulent. The meningococci can be seen in stained smears of cisternal fluid or of the brain surface, and they can be recovered usually in pure culture from these sources.

This infection usually remains localized, and in such cases the possibility that the meningococci which are recovered represent survivors of the inoculum must be considered. But sometimes the meningococci invade the blood stream, causing a generalized infection, and they can be recovered from the blood in pure culture. In such cases true infection can not be doubted. It is only very virulent strains which can produce such infection, and many recently isolated strains are without effect when given in the amounts used in these experiments.

Nonvirulent and heat-killed meningococci can also produce meningitis in guinea pigs when injected intracisternally in sufficiently large numbers. Such meningitis is not an infection and the microorganisms can not be recovered at autopsy. This toxicity of meningococci, whether killed or living, has been recognized for many years, and has been responsible for the general belief that meningococcus infection could not be produced in small laboratory animals, but that their death was due wholly to intoxication by the massive doses of meningococci given. Meningococci became so quickly attenuated in the laboratory that very little work was done with strains that possessed real virulence. If a culture had lost its virulence it made little difference whether it were killed or living in its effect upon the animals to which it was given.

Berkefeld filtrates of heavy suspensions of meningococci in Ringer's solution also produced meningitis when injected intracisternally into guinea pigs. Meningococci autolyze very rapidly, and it is uncertain whether the toxicity of these filtered washings is due to dissolved bacterial substance or to some substance washed from the outside of the cells. In either case, a small amount of the filtrate represented a very large number of meningococci.

Broth culture filtrates of certain strains of meningococci likewise produced meningitis on intracisternal injection. Relatively few

strains yielded strongly toxic filtrates but these few were consistent in their toxin production, though some lots were less potent than others. The "toxin"-producing property of meningococci was found to be less transient than virulence and was always associated with pellicle formation in the broth cultures.

Meningitis in guinea pigs, as described above, was not due to mechanical irritation or to the mere presence of foreign material, as was shown by intracisternal injections of broth, salt solution, and india ink, as well as filtrates of nontoxic strains and suspensions of other kinds of killed bacteria.

The clinical and pathological pictures that were found in the meningitis produced by living and killed cultures and by filtrates were essentially the same. This was to be expected, since meningitis is an inflammation of the meninges, and as such would have the same symptoms and pathology resulting from any cause which would produce a purulent exudate.

CONCLUSIONS

It appears that meningococcus meningitis in guinea pigs may be either (*a*) an infection or (*b*) an intoxication.

True infection has been found to occur only when strains of meningococci of marked virulence were used. Meningococci could be recovered from the cisternal fluid and brain surface, and sometimes from the heart blood.

Nonvirulent and killed meningococci caused meningitis when large numbers were given. Death in such cases was probably due to an intoxication.

"Toxins" produced by Ferry's method in broth cultures of certain strains of meningococci could also produce meningitis in guinea pigs.

In both infection and intoxication the clinical and histopathological pictures were the same.

Protocol 1

(Guinea pig D-3)

December 3, 1932: Weight, 275 gm; temperature, 38.5° C. Given 0.1 cc of living suspension of strain 433 (10 million cocci) intracisternally at 11 a. m. Lively after injection.

At 2 p. m. head was drawn to right; the animal shivered violently and constantly, and scratched his nose vigorously.

At 2:50 p. m. spasmodic jerking began. The head was held almost at a right angle to the body. The jerking became more violent, finally convulsions ensued, and death occurred at 6 p. m., 7 hours after injection. Placed in icebox overnight. Autopsied 10:30 a. m. on December 4, 1932. Stained smears from brain surface showed abundant polymorphonuclear leucocytes. No cocci definitely identified.

Brain.—Fibrinopurulent meningeal exudate most marked on base of cerebrum, thalamus, pons, medulla, and all around midbrain. Little pus in third ventricle purulent infiltration of choroid plexus of fourth ventricle. patchy round cell

infiltration of plexus of both lateral ventricles, marginal leucocyte infiltration of brain substance in pons and occipital cortex.

Diagnosis.—Acute purulent leptomeningitis.

Protocol 2

(Guinea pig J-1)

August 24, 1932: Weight, 225 gm; temperature, 40.0° C. Given 0.2 cc of living suspension of strain 479 (20 million cocci) intracisternally at 10 a. m. Well and lively after injection.

At 3 p. m. much prostrated; lost use of hind legs completely. Died at 6:30 p. m. Kept in icebox overnight. Autopsied 11:30 a. m. on August 25, 1932.

There was very little cisternal fluid. Smears showed disintegrated leucocytes and cocci chiefly in pairs. Pure culture of a group I–III meningococcus (strain 479 was of group I–III) was obtained.

Brain.—Pus in ventricles, foci of purulent infiltration in walls of lateral ventricles, floor of fourth ventricle and sides of third ventricle. Purulent thrombosis of scattered intracerebral vessels with purulent infiltration of surrounding brain tissue. Scattered small hemorrhages in brain stem. Diffuse purulent meningeal infiltration, more marked basally and in major fissures, less and partly lymphoid over convexity, accompanied by purulent infiltration of brain substance on base of cerebrum and pons.

Diagnosis.—Acute purulent meningitis.

Protocol 3

(Guinea pig H-15)

August 22, 1932: Weight, 230 gm; temperature, 38.0° C. Given 0.2 cc of living suspension of strain 487 (10 million cocci) intracisternally at 11 a. m. Animal well and lively after injection.

August 23, 1932: Weight, 220 gm; temperature, 36.0° C. Completely prostrated, paralyzed posterior extremities, apparently *in extremis.*

August 24, 1932: Marked opisthotonos. Tetanic spasms when touched or jolted.

August 25, 1932: Died at 8 a. m. Autopsied 9 a. m.

Smears from cisternal fluid showed monocytes, disintegrated polymorphonuclear leucocytes, and abundant gram-negative cocci.

Brain.—Marked pericellular edema and cell hydrops in cortex, tigrolysis in stem ganglia, empyema of lateral ventricles with softening, hemorrhages, small abscesses and purulent infiltration of adjacent brain tissue, pus in third and fourth ventricles, purulent meningeal infiltration especially on base and in major fissures.

Diagnosis.—Acute purulent meningitis.

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SERUM STUDIES IN EXPERIMENTAL MENINGITIS

Lack of Protection for Rabbits and Guinea Pigs[1]

By SARA E. BRANHAM, *Senior Bacteriologist*, and ANNA M. PABST, *Junior Bacteriologist*,[2] *United States Public Health Service, National Institute of Health*

It has seemed as though the greatest obstacle in the way of satisfactory evaluation of therapeutic antimeningococcic serums was the lack of success in producing meningococcus infection with dependable regularity in the smaller laboratory animals.

In August 1932 we described meningitis in rabbits (*1*) following intracisternal injections of recently isolated meningococci.

In December of the same year we reported, briefly, similar experimental meningitis in guinea pigs (*2*). A more detailed description of meningitis in guinea pigs is contained in a recent report (*3*).

In these animals, meningitis may be a true infection when highly virulent strains of meningococci are used, or an intoxication when large amounts of cultures of lower virulence are used. Meningitis due to "toxins", possibly, though not certainly, of a different nature, is produced by injection of broth culture filtrates of some strains of meningococci.

In the present paper we wish to report some of our studies on the effect of antimeningococcic serums on meningitis in both rabbits and guinea pigs.

To produce meningitis, two types of materials were used: (*a*) Newly isolated virulent cultures of meningococci and (*b*) toxic Berkefeld filtrates of 6-day broth cultures prepared after the method of Ferry (*6*) from several older strains which had been found to produce filtrates of marked toxicity. Most of the cultures were of strains which had been isolated only a few days previous to the experiments and had been shown to have unusual virulence for mice.

I. EFFECT OF SERUM WITH VIRULENT CULTURES

Eighteen-hour rabbit-blood agar slant cultures were suspended in Ringer's solution of pH 7.0–7.4. These suspensions were further diluted to correspond in density with standard suspensions of silica (*4*) so that the approximate number of micro-organisms injected was known. The number of meningococci injected was usually from 10,000,000 to 100,000,000, depending on the virulence of the strain. The volume was 0.2 to 0.3 cc. Injections were made intracisternally under ether anesthesia.

The 17 strains used in these serum studies were, on the whole, more virulent than those used in the earlier studies, and there was a tendency for the meningitis produced to follow the form especially

[1] Read before the Society of American Bacteriologists in New York City, Dec. 26, 1935.

[2] Miss Pabst died on Dec. 25, 1935, of meningococcus septicemia acquired in line of duty.

characterized by spasticity, opisthotonos, and hyperirritability (*1*) (*3*). With this form the animals frequently lived several days, and it was possible to follow the course of the disease in the rabbits by daily intracisternal puncture. Very cloudy fluids under increased pressure, and with cell counts of more than 100,000, were usually obtained. Differential cell counts with these spinal fluids showed a great preponderance of polymorphonuclear leucocytes. Intracisternal fluid was not easily obtained from guinea pigs.

The serum used in the experiments with living cultures included 12 polyvalent antimeningococcic horse serums from 6 different sources, 3 antitoxins (horse), and 5 normal horse serums.

During the earlier of these experiments the serum and culture suspension were mixed together and then injected intracisternally. The number of meningococci to be given was determined by preliminary experiments in which a constant volume of inoculum containing varying amounts of culture suspension was injected. An amount that would cause the death of at least 3, and preferably 4, out of 4 guinea pigs in 48 hours was taken as a standard dose, and this amount was mixed with 0.2 cc of serum to give a final volume of 0.3 cc. In some of the experiments with large rabbits, greater amounts of serum were used, but the total volume injected never exceeded 0.5 cc for these animals and 0.3 cc for guinea pigs. Guinea pigs were injected in quadruplicate and rabbits in duplicate or triplicate. Similar groups were given culture with normal serum, and also culture alone, to check the virulence of the strain used. Thus, a large number of animals was necessarily used. In experiments with antitoxin and with the usual polyvalent antimeningococcic serums, all made in horses, it was evident that there was no protection afforded by the amounts of serums given by this route. Appreciably larger quantities given intracisternally would cause an undesirable sudden increase in intracranial pressure.

The addition of fresh guinea pig complement to the serums before injection had no noticeable effect.

The effect of the horse serums alone when given intracisternally was studied. Here, polyvalent antimeningococcic serum, antitoxin, and normal horse serums were included. Apparently, some horse serum in itself has considerable toxicity for rabbits and guinea pigs when given by this route, and a meningitis often resulted which was similar clinically and histologically to that produced by the suspensions of meningococci.[3] This toxicity had no relation to the preservatives used in the serums, but is a property of the horse serum itself; guinea pigs were given, intracisternally, the same concentra-

[3] The brains of 44 guinea pigs that had received serum, serum+toxin, and serum+meningococci were examined histologically by Passed Asst. Surg. J. G. Pasternack. The findings were essentially the same as those which were described in detail in a former report (*5*).

tions of the preservatives that are used in the serums, with no ill effects. None of the normal rabbit and guinea pig serums tested showed any toxicity.

Since it was shown so clearly that horse serum is likely to be toxic when given intracisternally, and since the amount of any material that can be given by that route is very limited, it was decided to try the effect of injecting the serum intravenously, thus giving larger amounts. In some experiments the serum was given immediately preceding or following the intracisternal injection of the culture; in others, the serum preceded the culture by two hours or more. This intravenous administration of serums gave very little evidence, if any, of protection. There was no constant difference in effect between the immune serums and normal horse serum. A somewhat larger number of animals survived among those receiving the antitoxins, but the difference was not great enough to be significant.

Protection experiments in which amounts of serum as great as 4 to 6 cc were given intraperitoneally were entirely negative.

Most of the animals in which the meningitis was fatal died within 24 hours; but there were quite a number, especially among the rabbits, in which the disease ran a longer course. In some of these animals it was thought that frequent injections of serum might result in improvement, whereas a single initial injection had seemed useless. In a number of rabbits this course was followed, the serum being given intravenously daily, or twice daily, usually following the cisternal tap. These subsequent injections of serum not only failed to produce improvement, but actually seemed to hasten the death of the animals.

Although it has been relatively easy to produce acute purulent meningitis in rabbits and guinea pigs by intracisternal injections of virulent meningococci, it has not been possible to prevent or cure it by intracisternal, intraperitoneal, or intravenous administration of either the usual polyvalent antimeningococic serums or of antitoxins.

II. EFFECT OF SERUMS WITH TOXINS

The "toxins" used in these experiments were sterile Berkefeld N filtrates of meningococcus cultures grown for 4 to 6 days in a special broth after the method of Ferry (*5*). Not all strains of meningococci yielded filtrates of equal toxicity; they varied widely in this respect. A few strains proved to be consistently good "toxin-producers." This quality was always associated with the formation of a pellicle on the surface of the broth culture. Toxic filtrates prepared from 3 strains were dispensed, without preservative, into ampuls, which were sealed and stored at 3 to 5° C.

These toxins were titrated by intracisternal injection into guinea pigs. A standard fatal dose was taken as the least amount of the

toxin that would regularly kill, within 48 hours, 3 out of 4 guinea pigs weighing 200 to 250 grams. Not all of the filtrates could be titrated satisfactorily, but some gave very consistent results. Toxic filtrates 479A and 198A were chosen for the following experiments, and with both of these toxins, 0.2 cc of 1:2 dilution was taken as the standard dose.

The serum included in this study with toxins comprised 2 polyvalent horse antitoxins, 6 monovalent rabbit antitoxins, 13 polyvalent antimeningococcic serums (horse), 3 normal horse serums, and 3 normal rabbit serums. It was at this point in our studies that we began to realize that many horse serums were, in themselves, toxic for guinea pigs and rabbits when given by this route. Hence, each serum used was tested for toxicity by giving it alone to a series of 4 guinea pigs.

In the first three experiments the serums under study were added to the toxin, and the mixture was injected intracisternally, the volume injected being 0.2 or 0.3 cc, depending on the amount of serum used. In three experiments, 5 serums (1 antitoxin, 3 polyvalent antimeningococcic serums, and a normal horse serum) were tested for protection against toxin 198A when the toxin-antitoxin mixture was injected intracisternally. These 5 serums had been found to be practically nontoxic in themselves; and it was felt that any protective action which they possessed would, therefore, be evident in these experiments. In the first 2 of the 3 experiments, there was no evidence of protection. The two serums used in the third experiment had been concentrated, and there was apparently some protection in two groups of guinea pigs receiving mixtures of these serums with the toxin, though such results were obtained inconstantly.

As in the experiments with living cultures, the lack of evidence of definite protection by immune serums when given intracisternally led us to try other routes of administration. The toxin was given intracisternally, as before, and the serum injected intraperitoneally. No serum seemed to be of any value, whether the amount given was 1 cc or 4 cc.

These studies were followed by others in which the serums were given intravenously. The serum was given by this route immediately after the intracisternal injection of the toxin. Table 1 shows the results obtained in 3 experiments done several days apart with the same toxin and antitoxin. These experiments illustrate well how inconstant any protection offered seemed to be. The results in experiment 2 are almost dramatic, but experiments 1 and 3, done with the same material, show much less favorable results. However, in all three experiments the mortality among the guinea pigs receiving antitoxin intravenously was somewhat less than that among animals given the usual antimeningococcic serums by this route. This advantage for the antitoxic serums over the antibacterial is suggested again with

antitoxins AR7 and AR5 (both made in rabbits) in the experiments summarized in table 2, in which 9 polyvalent antimeningococcic serums (3 of them concentrated) and 4 antitoxins were tested simultaneously. The rabbit antitoxins were monovalent, and were specific for the toxin used in these experiments.

TABLE 1.—*Three experiments in which the same antitoxin was given intravenously to guinea pigs which had received the same toxin intracisternally*

Experiment no.	Number of animals	Amount of toxin 198A, 1–2	Amount of serum AH2	Effect	
				Died	Survived
		cc	*cc*		
I	4	0.2	1	1	3
	4	.2	2	2	2
	4	.2	----	3	1
II	4	.2	.5	1	3
	4	.2	1	1	3
	4	.2	2	0	4
	4	.2	4	0	4
	4	.2	----	4	0
III	4	.2	.5	3	1
	4	.2	1	1	3
	4	.2	2	3	1
	4	.2	4	1	3
	4	.2	----	3	1

TABLE 2.—*The effect of polyvalent antibacterial serums and antitoxins when given intravenously to guinea pigs which had received toxin intracisternally*

[Date: Aug. 13, 1933]

Number of animals	Amount of toxin 198A	Serum intravenously	Amount	Effect	
				Died	Survived
	cc		*cc*		
4	0.2	----	----	3	1
4	.2	BH16	1	3	1
4	.2	BH16	2	2	2
4	.2	BH17	2	2	2
4	.2	BH18	2	1	3
4	.2	BH19	2	3	1
4	.2	AH2	2	2	2
4	.2	AR7	2	1	3
4	.2	AR5	2	0	4
4	.2	BH20	2	3	1
4	.2	BH21	2	2	2
4	.2	BH22	2	4	0
4	.2	BH23	2	3	1
4	.2	BH24	2	2	2
4	.2	AH4	2	3	1

In our studies, rabbits and guinea pigs, and also mice, were found to be highly resistant to the meningococcus toxic filtrates when they were given intravenously, some of the rabbits being able to receive as much as 6 cc by this route without ill effect, though others succumbed to smaller amounts. The toxin seemed to be chiefly neurotropic in its action. Thus, studies on intravenous injection of toxins and toxin-antitoxin or toxin-serum mixtures could not be made satisfactorily.

A word should be added about the stability of the toxins used in these studies. Storage temperature was 3° to 5° C. At first all ampuls of any given lot seemed to be exactly alike, and the standard dose remained constant. After a year had elapsed it was noticed that individual ampuls varied in strength. Titrations were made at frequent intervals, and a control group of animals, receiving toxin only, was included, as usual, with each experiment. Some of the lots of toxin are now more than 5 years old, and at least one-half of the ampuls recently tested seem to be as toxic as they were when first made, though others have become very weak.

These studies made with toxin in guinea pigs do not indicate that dependable and regular protection is afforded by any of the serums studied when given by any of the routes used.

III. BACTERIOLOGICAL STUDIES

Most of the animals that died were autopsied and cultures on blood agar were made from the surface of the brain and from the heart blood. Among those animals that were given living cultures, meningococci were recovered more regularly from guinea pigs than from rabbits, though they were obtained occasionally from rabbits also. In some guinea pigs, meningococci were obtained from the heart as well as from the brain, but more commonly they were found only in those cultures made from the brain. They were never obtained from the heart of any rabbit. Meningococci were recovered as often from those animals that received serum as from those that did not, but more often when the serum was given intravenously or intraperitoneally than when it was given intracisternally. Meningococci were recovered from about one-fourth of all of the animals given serum intravenously and from about one-sixth of those given serum intracisternally.

Cultures were also made from the dead animals given only toxin or serum intracisternally in order to be sure that they did not die of some intercurrent infection. As a rule, cultures from animals dying within 48 hours after injection were negative for all bacteria, whereas those from animals that succumbed after a longer period of time were more likely to show the presence of secondary invaders. By far the most common of these was a small Gram-negative rod of the genus *Pasteurella* and a large Gram-positive coccus. These were infrequent.

DISCUSSION

Acute purulent meningitis can be produced in rabbits and guinea pigs by the intracisternal injection of cultures of meningococci which are sufficiently virulent. Meningitis which is essentially identical

both clinically and histopathologically is produced by similar injections of Berkefeld filtrates of 4–6-day broth cultures of some strains of meningococci. In the hands of the authors these toxin-producing strains have always proved to be of serological group I–III.

Attempts to protect rabbits and guinea pigs against meningitis, whether due to living cultures or to toxins, by means of serums, have not met with success. Twenty-four polyvalent antimeningococcic serums (horse) from 8 sources, 4 polyvalent antitoxins (horse), 13 monovalent antitoxins (rabbit), and a number of normal serums, including horse, rabbit, and guinea-pig, have been given to animals which had received toxin or living cultures. The toxins and cultures were given intracisternally, whereas the serums were given intracisternally, intraperitoneally, intravenously, or by a combination of routes.

Slightly better results were obtained with the antitoxins than with the usual unconcentrated antimeningococcic serums, but the difference was not great enough to be of definite significance, and the apparent protection was inconstant. There is some evidence that concentrated serums may offer some protection. The amount of serum that can be given intracisternally without causing pressure symptoms is very small. Many horse serums are toxic in themselves when given to rabbits and guinea pigs by this route.

When living cultures are given intracisternally, the infection usually remains fairly well localized in the meninges; invasion of the blood stream does occur not infrequently in guinea pigs, but has not been observed by us in rabbits. To what extent the toxin becomes disseminated throughout the body when given intracisternally is not known, but relatively large amounts of it seem well tolerated by rabbits and guinea pigs when it is given intravenously. There is no evidence that the relatively large amounts of serum given intravenously have come in contact with the inflamed meninges. Thus, it seems that the meningitis experimentally produced in rabbits and guinea pigs by the meningococcus or its toxic products does not offer a satisfactory basis for the study of the comparative therapeutic value of serums, whether antibacterial or antitoxic, by the methods which we have used. In this respect, our results are at variance with those of Zdrodowski and his coworkers (*6*).

CONCLUSIONS

Experimental meningitis in rabbits and guinea pigs has not provided a basis for the study of the therapeutic value of the antimeningococcic serums or meningococcus antitoxins included in our studies.

Meningitis was easily produced in these animals, but they were

not protected to any appreciable degree by the serums when they were administered intracisternally, intraperitoneally, or intravenously.

Some of the experiments have suggested that with more perfect methods of concentrating the serums, better protection may be obtained.

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Addendum

The experiments described in the two foregoing papers were done several years ago before the discovery, by Nungester and Miller, of the mucin technique for enhancing the virulence of meningococci.

Since the completion of our work, reports on experimental meningitis in rabbits or guinea pigs have been made by Maegraith, Ferry, Monteiro, Zdrowdowski and Golinewitch, Pawlow, Puschnowa and Cryjanowskaja, and Petrie. The conflicting results obtained by some of these workers may possibly, in the light of our foregoing papers, be partially explained.—*S. E. Branham.*

PLANS OF THE CHILEAN GOVERNMENT FOR IMPROVING THE NUTRITION OF THE PEOPLE[1]

By Eduardo Cruz Coke, M. D., *Minister of Health, Social Security, and Welfare of Chile*

From the researches of a study mission of the Health Committee of the League of Nations,[2] from studies made in Chile by Drs. Arturo Mardones, Luis Calvo Mackenna, Carlos Garcés, Ramón González, Julio Santa María, Luis Toro Genkel, Jorge Mardones Restat, and my own investigations, the following deficiencies are seen to be characteristic of Chilean nutrition:

(1) Malnutrition of an important part of the infant population, with its effects on physical development; (2) insufficient production in Chile of foods which the Health Committee of the League of Nations considers protective (milk and its derivatives, meat, eggs, green vegetables, and fruits); (3) phosphorus and calcium deficiency in foods produced in some parts of the country, especially phosphorus; (4)

[1] From an address to the National Council on Nutrition, Chile, Feb. 18, 1937. Translated from the *Boletín de la Oficina Sanitaria Panamericana*, July 1937

[2] Studies made in 1935-36 at the request of the Chilean Government. Results as yet unpublished.

lack of meat and proteins in the diet of an important part of the population; (5) little variety in the habitual diet.

The Chilean people obtain their food fundamentally from wheat and potatoes, in an unvaried diet. The consequences of the deficiencies noted are seen in the poor development of the children, with its evident dangers for the future of the race. Therefore the Ministry of Health is centering its attention on the child, whose height, development, and health depend principally on its nutrition, as was shown by the studies of Dr. Jorge Mardones Restat and collaborators, who analyzed the relationship between these characteristics and the social conditions of a group of children in the primary schools of Santiago.

Before describing a tentative plan of action, it would be well to mention the agencies of the State which may be utilized in any measures affecting production and consumption.

The Government has three important departments regulating production: The Farm Credit Board, the Agricultural Exports Board, and the Farm Settlement Board. In addition, it acts through other public departments in encouraging the fishing industry, and in bringing meat from Magallanes [3] to the market centers of the Central Region.

The Government may also act on consumption through the Central Subsistence Bureau and the warehouses of the Department of Social Security to influence the prices of foods. It may educate the public on proper nutrition through the various branches of the public school system, as well as through the Army and Navy and the Department of Nutrition of the National Department of Health. All these organizations have, until now, acted independently, without the coordination which would have permitted regulating production in accordance with consumption. The coordination of these bodies has now been entrusted to the National Council on Nutrition, made up of experts representing both producers and consumers.

The Farm Credit Board should direct its activities toward the making of loans at low interest rates to those agricultural enterprises which the Government, as represented by this Council, may consider, at any given moment, of most importance in relation to the national food supply.

The Agricultural Exports Board, without abandoning its function of stimulating the exportation of farm products, should fix the export quotas in proportion to the necessities of the moment, as suggested by this Council. It must be considered as a fundamental principle that the primary function of our national agriculture is to produce the necessary foods for consumption by our population, and only secondarily to export the surplus. Intensive development of our

[3] Magallanes is a remote province at the southernmost end of Chile and is the center of the cattle industry.

farm territory will permit the fulfillment of both functions. This principle does not imply that there should not be produced a certain amount to be exported advantageously with respect to the world market, but in such a case there must be worked out some means to supply the nutrients which, by reason of this exportation, are lacking.

The Farm Settlement Board shall, through the gradual division of land ownership, secure a spontaneous change from extensive to intensive development of land, obtaining thus an increase in the yield. Its supervised colonies and its recommendations to landholders should constitute factors of prime importance in the orientation of agricultural production in accordance with the necessities of the country.

But production is linked not alone with this type of activity. Food does nothing more than transport from the earth to man specific materials. No aliment contains what the earth does not give it. The latest statistics show that the average yield of agricultural production tends to diminish, and that the level of production is being maintained only by the extension of cultivated territory. This is the consequence of the impoverishing of that which a country should care for above all—the soil from which it is nourished. The indolence of many farmers who do not return to their fields by adequate fertilizing what they take from them may be considered as a potential, if not immediate, peril. There is needed, then, a policy of distribution of fertilizer which will result in an increased farm yield and in a better quality of vegetable foods produced.

With regard to the stimulation of the consumption of protective foods, the Central Subsistence Bureau and the Department of Social Security, through their warehouses, should cause the burden of costs to fall on the least useful foods, in order to permit a minimum price for those the consumption of which it is most necessary to stimulate. The determination of the degree of utility of an aliment in relation to its physiological value and the state of the national production shall be undertaken by this Council, which shall guide both departments in this regard.

The collaboration of the various branches of the educational system is of first importance. It is no exaggeration to say that the progress of a country is most greatly influenced by a system which teaches the child first to live correctly—eat, live, dress, and work correctly—rather than to read rapidly. Practical teaching of correct nutrition may be accomplished in part in the primary school through the school lunch, prepared in accordance with the recommendations of this Council. In boarding schools, barracks, and on ships a similar result will be obtained by rational nutrition, developing habits likely to be followed when the individual leaves these places.

The expending of funds which the state or municipality contributes to the upkeep of children's institutions should be in accordance with standards fixed by this Council.

About 50 percent of the population of Chile lives in rural districts. Contrary to what might be thought, an important proportion of this group, especially the children, is very badly nourished, to such a point that a great deal of our tuberculosis finds its focus in the rural population, and, likewise, other conditions develop when the organic defenses are weakened. There are, then, urgently needed measures which will assure to this population the minimum necessary, not for mere existence, but to secure the most modest standards of bodily development.

To this end, this Ministry has already contacted numerous farmers, and wishes, through this Council and the agricultural societies, to bring to the attention of the farmer the standards to which his public duty requires him to adhere, with regard both to his tenants' welfare and to seeing that his farm contributes its part toward the public need.

For this there has been undertaken the creation of an Association of Farm Proprietors, under the sponsorship of the Ministry of Health and of this Council, to see that the family of the tenant has available the proper bases for nutrition and uses them in the manner most suitable to life and health.

The primary necessities which must be taken care of, for the future of our people, may be summarized in two points: It is necessary that each child of the tenant have a half liter of milk daily, up to the age of 8 years, and a half kilo of meat or legumes weekly to the age of 15 years; and each nursing mother must have 1 liter of milk per day.

The Government does not deem it expedient to go more deeply into the particular economy of agricultural development at this moment, and has set up these standards with the idea that the administrator shall be responsible for this minimum program, compatible with the poorest agricultural returns, there being no excuse for failure to comply with this plan. The moral promise contracted by the administrator, within the organization established, will be supervised by the members of the association, and, on the part of the Ministry, by social workers or whoever may replace them. Each patron or administrator will know what means to take to solve the problem within his capacities. No one shall be asked to do the impossible, but all necessary influence will be exercised to see that no one fails to do what he is in a position to accomplish. The Government will request this cooperation from agriculture and is sure of receiving it. A refusal to cooperate would justify a similar refusal by the Government to furnish the aid which is being constantly requested of it.

The difficulties and objections which a few have raised against a system such as this, entailing no direct penalties, are not valid except

to those who are unaware that there do exist in this country moral values and a desire to serve in a much greater proportion and intensity than superficial examination reveals. The experience obtained in the province of O'Higgins shows that intelligent competition, timely propaganda, and social pressure can in many cases accomplish more than laws carrying heavy penalties. I do not believe that the Farm Proprietors Association to which I refer will fail to secure the fulfillment of the minimum requirements of 500 grams of milk daily and 500 grams of meat or legumes weekly, especially since this is no vague promise to contribute to an ill-defined measure of well-being, but a precise obligation expressed in weights and measures.

The most serious problem confronting the Ministry of Health in the solution of the nutritional problems requiring immediate consideration is the gradual decrease in the production of milk, since our country already produces it in a quantity very much below its minimum necessities (from one-fifth to one-tenth as much per inhabitant in relation to the European, American, and Australian production—Switzerland, 600 liters per inhabitant per year; France, England, and Germany, 300–400; Chile, 40). If it is remembered that milk is the principal material for the building of a race, irreplaceable by any other food in nearly all cases, that it is the most important of the protective foods, and that according to our own studies and those of our coworkers it has, furthermore, an inhibitory action on precocious sexual development, the primary interest of the Department of Public Health in its maximum consumption will be understood.

All attempts at decreasing infant mortality are useless if the child and the nursing mother do not have sufficient milk, and all moral education of children becomes purposeless if through lack of milk puberty is advanced and, with this, precocious differentiations resulting in an organic and psychological unbalance.

The Government has already suggested measures to stimulate consumption and production of milk, which the Council will carry on; these include the school lunch, the contribution being almost entirely in milk, and perfecting of the relations between production and distribution, involving pasteurization plants.

Investigation of the causes of decreased production of milk has shown that it is due to the fact that the economic return from milk production has not increased in proportion to that from other farm products which, furthermore, are easier to produce and do not necessitate control and regulation. Means are being studied of increasing production not only through increased consumption, the state itself being a large purchaser for the school lunch, but also through lending at low interest the extra capital needed by the milk industry. The installation of milk drying or condensing plants in the grazing regions

of the South, permitting utilization of it in periods of great abundance, is another measure which may be established.

On the initiative of the Ministry of the Treasury, the Government has resolved on the use, in part, of meat from Magallanes, and is considering the establishment of a refrigerating system in the principal ports and centers of consumption which will also serve as a solution of the problem of preserving fish, permitting larger exploitation of the fishing industry, and of the preservation of fruit for exportation. This year there will be brought in from 2,500 to 3,000 tons of frozen meat at a price within the reach of modest salaries, a quantity representing the minimum capacity of the existing refrigerating plants. The Government is also disposed to use in increasing the supply of meat a system formerly used with success—that of loans to the raisers of small stock (sheep and hogs) in other sections of the country.

The deficiency in phosphorus, producing dental caries, bone deformities, growth disturbances, and so on could be solved over a number of years by means of phosphate fertilizers. The Ministry of Health has decided that the most simple and practical manner of solving this important problem immediately is by adding to common salt a quantity of phosphorus determined as immediately assimilable, and the corresponding law has been issued, so that the people of Chile will receive, through salt in their food, about 0.50 grains of assimilable phosphates.[4]

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[4] Text of the law on phosphorus, Santiago, Feb. 23, 1937:

Whereas after various studies the conclusion has been reached that among the principal deficiencies in our nutrition is the small concentration of mineral salts; that this deficiency results in the frequency of dental caries and bony disturbances in growth; that it is urgently necessary to arrest this poverty which results in an increase in morbidity and a lowered resistance of our population, which does not have access to a diet compensating these deficiencies; that among the most important of these materials as regards the lack of it and its value is phosphorus; that the most practical, economical, and rapid manner of securing to all the inhabitants of the country a supply of this substance in quantities which will make up for the deficit is to add it to a basic food element of wide and varied application; that common salt (kitchen salt, table salt) combines all these characteristics. By virtue of the authority vested in me by article 166 of the Sanitary Code in force,

I hereby decree that—

1. Salt for food purposes shall contain the quantity of 4 percent of acid sodium phosphate.

2. Salt plants, establishments, or factories engaged in the crystallization, extraction, or preparation of common salt, and the retailers of it, may not distribute salt for food purposes if it does not contain, in the indicated proportion, the product named in the previous article.

3. The enforcement of this Decree is in charge of the National Department of Health.

4. Any actual or legal person failing to comply with the regulations set up in the articles 1, 2, and 3 of this Decree shall be punished with a fine of from 50 to 1,000 pesos, and in addition the merchandise which does not comply with the requirements here established may be confiscated. The same penalties shall apply to anyone who, in the preparation of food products, uses salt which fails to comply with the provisions of Article 1 of this Decree.

5. Salt used exclusively for industrial purposes, such as that used in refrigerating plants, freezers, soda factories, and for salting hides and tripe, where food products are not produced, or which is not used in food products, is exempt from the above provisions.

To be exempt, producers or sellers of common salt shall require, and industries shall secure from the National Department of Health, special purchase permits, stating in the application the salt factory or establishment where the salt is to be purchased, declaring that it will not be used for food purposes, indicating the quantity purchased, and obligating themselves not to resell the salt, even in part, without the necessary permission.

6. This Decree shall be in force 120 days after its publication in the Diario Oficial.

(Signed) ALESSANDRI E. CRUZ COKE.

The stimulation of the division of farm lands will permit a greater production of green vegetables and fruits (protective foods), as already observed in Chile wherever the Farm Settlement Board had available the economic possibilities of accomplishing it. This development will be intensified, and the Council will advise as to the type of production to be required by the Department.

The problem of fertilizer distribution may be solved, in part, after further study, by requiring for the exportation of all foods involving a considerable extraction from the soil of nitrogen, phosphorus, and calcium, a certificate showing that the farmer has returned to his soil, by adequate fertilization, the elements which the exportable crop has extracted. This will, over a period of time, benefit the farmer himself, since he will not only obtain a larger immediate yield, but his land will not become impoverished.

The high consumption of wheat is an indication of the lack of other foods of better quality such as those of animal origin and the legumes. Measures to stimulate the consumption and production of beans are justified by the consideration that the bean crop has decreased from a million quintales in 1933, with a consumption of 800,000, to 700,000 in 1936 with a consumption of 469,000.[5]

The Ministry of Labor, by influencing prices or salaries through the Central Subsistence Bureau will try to see that the prices of foods are at a level at which they may be obtained by persons in the low-wage group in sufficient quantity for normal sustenance; the Ministry of Agriculture is revising its loan and export policies in accordance with the plans outlined above.

The Government, speaking through the Ministry of Health, believes that it has demonstrated with shocking frankness the precarious state of national nutrition; and having completed studies of the problem, it has begun to adopt all the measures within its power to bring about not only an immediate solution where possible, but also to establish the bases of a true policy of nutrition which shall embrace all pertinent departments of the State. The Government has tried to harm as few interests as possible, but considers the health of the people of first importance.

This Ministry has not referred, except in passing, to a problem undoubtedly linked with that of nutrition—that of wages, a problem which another department of the State is attempting to solve in accordance with the minimum needs of the people.

With regard to the Council on Nutrition, its influence and possibilities for accomplishment depend not on the powers given it but on the zeal which its members display for the public welfare. The

[5] A quintal is approximately 100 pounds.

country possesses enormous resources unknown to the outsider. The Government realizes this, and it is with great hopes that the work of this Council is begun.

ADDING SODIUM ACID PHOSPHATE TO TABLE SALT TO CORRECT PHOSPHORUS DEFICIENCY

With reference to the addition of acid sodium phosphate to table salt for the purpose of correcting a dietary deficiency of phosphorus, Dr. W. H. Sebrell, of the United States Public Health Service, doubts the efficacy of such a measure as that provided in the decree quoted in the preceding article, in which it is required that all salt used for food purposes shall contain 4 percent of acid sodium phosphate. The following is an excerpt from a memorandum on the subject recently prepared by Dr. Sebrell:

Sollman states that the phosphate ion is poorly absorbed. The absorption depends, partly at least, upon the amount of calcium and vitamin D present. However, disregarding such important considerations as the utilization of phosphorus furnished in this manner, and the advisability of increasing dietary phosphorus without increasing dietary calcium proportionately, the practical futility of attempting to make a diet adequate in phosphorus by adding sodium acid phosphate to table salt can be shown by a little simple arithmetic.

The average daily consumption of sodium chloride by adults has been variously estimated at from 10 to 20 grams per day. The average daily requirement is estimated at about 2 grams per day. From the available estimates of the amount of sodium acid phosphate proposed to be imported into Chile annually, it would appear that the Chilean consumption of sodium chloride is well within this range and possibly near the lower figure. However, we may base our calculations on the highly improbable maximum average daily intake of 20 grams of sodium chloride:

1 gram of NaH_2PO_4 (sodium acid phosphate) contains 0.224 gram of phosphorus.

If a 4-percent mixture is made with sodium chloride, every gram of the sodium chloride–sodium acid phosphate mixture contains 0.00896 gram of phosphorus.

On the basis of an average daily intake of 20 grams of the sodium chloride mixture, the average daily intake of phosphorus would be only 0.1792 gram.

Since an adult needs 0.88 gram of phosphorus daily for maintenance (Sherman), and the recommended intake per day per adult is 1.32 grams, and the fact that the requirement for children and pregnant and lactating women is even higher than this, it is obvious that the addition of a variable amount of phosphorus, which in all probability will not equal 0.1792 gram per adult per day and will more than likely be in the neighborhood of half of this figure and even less for children, will not be sufficient to correct any material deficiency in phosphorus existing in the population.

DEATHS DURING WEEK ENDED JULY 31, 1937

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended July 31, 1937	Corresponding week, 1936
Data from 86 large cities in the United States:		
Total deaths	7,248	7,095
Average for 3 prior years	7,988	
Total deaths, first 30 weeks of year	272,717	273,970
Deaths under 1 year of age	489	497
Average for 3 prior years	537	
Deaths under 1 year of age, first 30 weeks of year	17,229	17,148
Data from industrial insurance companies:		
Policies in force	70,091,298	68,393,465
Number of death claims	13,217	14,038
Death claims per 1,000 policies in force, annual rate	9.8	10.7
Death claims per 1,000 policies, first 30 weeks of year, annual rate	10.4	10.4

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Aug. 7, 1937, and Aug. 8, 1936

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936
New England States:								
Maine	----	5	----	----	----	33	0	0
New Hampshire	----	1	----	----	8	2	0	0
Vermont	----	1	----	----	----	----	0	0
Massachusetts	6	7	----	----	36	89	3	0
Rhode Island	----	----	----	----	1	1	0	0
Connecticut [4]	8	1	----	1	18	16	0	3
Middle Atlantic States:								
New York	20	17	[1] 3	[1] 2	151	223	9	11
New Jersey	2	1	----	6	73	50	0	1
Pennsylvania	17	13	----	----	257	73	10	5
East North Central States:								
Ohio	6	7	1	3	77	17	3	1
Indiana	2	8	----	4	33	1	2	3
Illinois	15	17	1	6	80	3	7	3
Michigan	7	4	----	----	68	8	1	2
Wisconsin	3	4	28	7	38	61	1	0
West North Central States:								
Minnesota	5	1	1	1	4	3	0	0
Iowa	2	3	1	2	6	3	0	4
Missouri	7	5	25	27	1	----	1	0
North Dakota	3	1	2	----	----	----	0	0
South Dakota	----	1	----	----	----	2	1	0
Nebraska	----	6	----	----	2	8	0	0
Kansas	1	6	----	1	7	----	1	0
South Atlantic States:								
Delaware	----	----	----	----	----	1	0	0
Maryland [3]	4	8	----	2	13	30	0	2
District of Columbia	3	1	----	----	3	7	2	0
Virginia [3] [4]	17	10	----	----	13	14	1	6
West Virginia	3	3	14	----	24	3	0	2
North Carolina	8	18	----	4	32	5	3	1
South Carolina [4]	8	1	42	32	34	5	0	0
Georgia [4]	17	8	----	----	----	----	3	2
Florida [4]	2	6	----	1	8	----	0	2
East South Central States:								
Kentucky	3	2	1	----	21	14	4	13
Tennessee [3] [4]	7	8	8	13	7	5	1	2
Alabama [4]	11	9	5	11	1	1	3	0
Mississippi [3] [4]	9	10	----	----	----	----	0	0
West South Central States:								
Arkansas	5	3	4	1	2	----	0	0
Louisiana [4]	9	5	10	6	2	5	1	0
Oklahoma [3]	5	4	1	11	6	1	2	0
Texas [4]	31	24	53	26	36	33	5	1
Mountain States:								
Montana [3]	1	1	----	----	3	1	0	2
Idaho	----	----	3	----	4	4	1	0
Wyoming	1	1	----	----	2	1	0	0
Colorado [3]	6	2	----	----	12	2	0	1
New Mexico	5	1	----	1	21	1	0	0
Arizona	----	3	11	7	1	28	0	0
Utah [3]	----	----	----	----	4	4	0	0
Pacific States:								
Washington	1	----	----	----	16	20	0	0
Oregon	1	----	8	4	7	6	0	1
California	21	17	7	8	21	67	2	2
Total	282	254	229	187	1,153	851	67	70
First 31 weeks of year	13,093	14,096	274,029	139,684	239,154	266,228	4,058	5,772

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Aug. 7, 1937, and Aug. 8, 1936—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936	Week ended Aug. 7, 1937	Week ended Aug. 8, 1936
New England States:								
Maine	13	0	2	4	0	0	0	1
New Hampshire	0	0	1	1	0	0	0	1
Vermont	0	1			0	0	0	0
Massachusetts	12	2	38	46	0	0	1	4
Rhode Island	1	0	2	5	0	0	0	2
Connecticut [4]	3	1	13	8	0	0	2	1
Middle Atlantic States:								
New York	17	8	71	111	0	0	18	13
New Jersey	3	0	12	14	0	0	7	4
Pennsylvania	11	3	126	111	0	0	25	22
East North Central States:								
Ohio	38	4	35	42	0	0	28	4
Indiana	7	1	24	23	2	0	6	7
Illinois	28	11	91	111	0	1	19	19
Michigan	14	3	89	76	0	0	5	6
Wisconsin	6	0	48	78	1	3	3	2
West North Central States:								
Minnesota	9	0	25	22	2	1	3	1
Iowa	4	1	26	27	3	1	4	3
Missouri	16	3	15	15	0	1	14	26
North Dakota	0	0	5	8	3	0	2	0
South Dakota	1	0	5	20	6	0	1	0
Nebraska	7	0	2	8	1	0	1	0
Kansas	13	0	23	31	1	1	15	4
South Atlantic States:								
Delaware	0	0	8	2	0	0	5	1
Maryland [3]	3	1	5	10	0	0	14	8
District of Columbia	0	0	1	1	0	0	2	1
Virginia [3 4]	4	2	8	15	0	0	57	35
West Virginia	12	2	18	9	0	0	15	11
North Carolina	4	2	25	13	0	0	25	45
South Carolina [4]	1	2	11		0	0	12	18
Georgia [4]	6	6	7	5	0	0	36	25
Florida [4]	0	0	2	2	0	0	4	3
East South Central States:								
Kentucky	9	4	20	7	0	1	43	42
Tennessee [3 4]	3	26	8	3	1	0	43	40
Alabama [4]	3	26	11	8	0	0	19	36
Mississippi [3 4]	8	12	4	5	0	0	16	17
West South Central States:								
Arkansas	21	0	3	5	0	0	37	15
Louisiana [4]	7	1	5	17	0	0	23	39
Oklahoma [5]	[6] 30	0	7	10	0	0	23	49
Texas [4]	58	0	31	17	0	1	113	87
Mountain States:								
Montana [3]	2	0	7	3	9	8	3	2
Idaho	0	1	6	7	6	1	0	1
Wyoming	2	0	5	9	0	2	2	0
Colorado [3]	2	0	12	11	0	0	0	2
New Mexico	0	0		5	0	0	2	7
Arizona	0	0		1	0	0	1	4
Utah [3]	0	0	4	4	0	3	1	1
Pacific States:								
Washington	1	5	19	18	5	0	11	3
Oregon	2	1	9	7	2	0	8	5
California	33	9	50	61	10	1	18	17
Total	414	138	939	1,016	52	25	687	634
First 31 weeks of year	2,485	1,174	163,175	176,932	7,847	5,863	6,813	6,146

[1] New York City only.
[2] Week ended earlier than Saturday.
[3] Rocky Mountain spotted fever, week ended Aug. 7, 1937, 9 cases, as follows: Virginia, 5; Tennessee, 2; Montana, 1; Colorado, 1.
[4] Typhus fever, week ended Aug. 7, 1937, 101 cases, as follows: Connecticut, 1; Virginia, 1; South Carolina, 1; Georgia, 62; Florida, 6; Tennessee, 1; Alabama, 13; Mississippi, 1; Louisiana, 2; Texas, 13.
[5] Figures for 1936 are exclusive of Oklahoma City and Tulsa.
[6] Delayed report of 10 cases included.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
March 1937										
Tennessee	33	37	1,539	10	86	13	1	85	0	9
July 1937										
Delaware					6	1	1	3	0	3
District of Columbia		29			109	2	1	14	0	17
Idaho	2	1	18		36		1	52	35	3
Iowa	7	15	2	5	38		7	117	111	14
Nebraska	3	5			33		20	40		4
Vermont		4			17		2	8	0	0

March 1937

Tennessee:	Cases
Chicken pox	232
Dysentery (amoebic)	3
Dysentery (bacillary)	2
German measles	101
Mumps	180
Ophthalmia neonatorum	4
Paratyphoid fever	1
Puerperal septicemia	1
Septic sore throat	5
Tetanus	1
Trachoma	12
Tularaemia	4
Vincent's infection	13
Whooping cough	158

July 1937

Chicken pox:	
Delaware	5
District of Columbia	18
Idaho	16
Iowa	57
Nebraska	15
Vermont	32

July 1937—Continued

Conjunctivitis:	
Idaho	1
Dysentery (bacillary):	
District of Columbia	1
Encephalitis, epidemic or lethargic:	
District of Columbia	1
Idaho	1
German measles:	
Delaware	1
Idaho	4
Iowa	4
Impetigo contagiosa:	
Idaho	1
Mumps:	
Delaware	6
Idaho	46
Nebraska	15
Vermont	149
Ophthalmia neonatorum:	
Idaho	1
Rocky Mountain spotted fever:	
Delaware	1
District of Columbia	1

July 1937—Continued

Rocky Mountain spotted fever—Continued:	
Idaho	5
Iowa	3
Septic sore throat:	
Idaho	8
Nebraska	1
Vermont	1
Trachoma:	
Idaho	8
Iowa	7
Tularaemia:	
Idaho	1
Undulant fever:	
Iowa	11
Vermont	5
Whooping cough:	
Delaware	77
District of Columbia	46
Idaho	55
Iowa	152
Nebraska	65
Vermont	79

PLAGUE IN GROUND SQUIRREL, BEAVERHEAD COUNTY, MONT., AND IN FLEAS FROM CHIPMUNKS, ORMSBY COUNTY, NEV.

Under date of August 3, 1937, plague was reported demonstrated in tissue from a ground squirrel (*Citellus elegans*) shot 3 miles south of Dillon, Beaverhead County, Mont. On August 4, 1937, plague infection was stated proved in a lot of 86 fleas taken from 52 chipmunks (*Eutamias frater*) shot 12 miles west of Carson City, Ormsby County, Nev.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 31, 1937

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	123	40	13	745	301	349	6	380	95	1,328	----
Current week [1]	85	21	6	671	285	283	7	340	65	1,401	----
Maine:											
Portland	0	----	0	1	1	0	0	0	0	3	17
New Hampshire:											
Concord	----	----	----	----	----	----	----	----	----	----	----
Manchester	0	----	0	----	2	0	0	0	0	0	17
Nashua	0	----	0	2	0	0	0	0	0	0	3
Vermont:											
Barre	0	----	0	1	0	0	0	0	0	0	3
Burlington	0	----	0	0	0	0	0	0	0	0	11
Rutland	0	----	0	0	1	0	0	0	0	0	8
Massachusetts:											
Boston	2	----	0	3	6	11	0	8	0	34	179
Fall River	1	----	0	1	1	1	0	0	0	3	20
Springfield	0	----	1	0	1	2	0	0	1	6	36
Worcester	0	----	0	2	5	1	0	2	1	5	----
Rhode Island:											
Pawtucket	0	----	0	0	0	0	0	0	0	0	11
Providence	0	----	0	6	3	2	0	1	0	18	55
Connecticut:											
Bridgeport	0	----	0	0	0	1	0	3	0	0	20
Hartford	0	----	0	6	1	0	0	3	0	4	31
New Haven	0	----	0	6	0	1	0	1	0	2	32
New York:											
Buffalo	0	----	0	7	7	0	0	4	0	37	131
New York	30	4	2	127	51	27	0	69	6	91	1,151
Rochester	0	----	0	2	1	1	0	0	0	13	50
Syracuse	0	----	0	10	1	0	0	1	0	0	31
New Jersey:											
Camden	0	----	0	2	0	1	0	1	1	4	24
Newark	0	----	0	5	0	1	0	4	0	29	81
Trenton	0	----	1	9	7	0	0	4	0	4	33
Pennsylvania:											
Philadelphia	0	----	0	0	5	12	0	21	6	64	387
Pittsburgh	1	1	0	43	14	7	0	9	0	62	148
Reading	0	----	0	4	0	1	0	1	0	0	18
Scranton	0	----	----	0	----	0	0	----	0	2	----
Ohio:											
Cincinnati	1	----	0	11	4	3	0	7	2	32	132
Cleveland	1	2	0	62	8	11	0	10	1	57	168
Columbus	0	----	0	26	1	4	0	3	0	37	88
Toledo	0	----	0	16	1	1	0	6	2	34	66
Indiana:											
Anderson	0	----	0	5	2	0	0	0	0	4	10
Fort Wayne	0	----	0	0	0	1	0	0	0	0	25
Indianapolis	2	----	0	5	6	1	1	5	1	26	90
Muncie	2	----	0	0	0	0	0	2	0	0	14
South Bend	0	----	0	2	0	0	0	0	0	0	11
Terre Haute	0	----	0	0	0	1	0	0	0	0	22
Illinois:											
Alton	0	----	0	1	0	2	0	0	1	0	5
Chicago	11	----	1	125	21	42	0	35	5	83	587
Elgin	0	----	0	0	1	0	0	1	0	1	13
Moline	0	----	0	0	0	1	0	0	0	9	6
Springfield	0	----	0	1	0	0	0	1	0	1	25
Michigan:											
Detroit	3	1	0	22	6	40	0	8	1	58	218
Flint	0	----	0	1	2	4	0	1	0	1	19
Grand Rapids	0	----	0	8	0	3	0	0	0	19	17
Wisconsin:											
Kenosha	0	----	0	0	0	1	0	0	0	0	8
Milwaukee	0	----	0	10	2	8	0	2	0	39	74
Racine	0	----	0	0	0	6	0	1	0	2	11
Superior	0	----	0	0	0	0	0	0	0	7	5

[1] Figures for Concord, N. H., St. Joseph and St. Louis, Mo., estimated; reports not received.

City reports for week ended July 31, 1937—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0		0	0	1	3	0	1	0	1	20
Minneapolis	0		0	0	6	3	1	0	0	10	83
St. Paul	0		0	0	4	1	0	3	0	47	53
Iowa:											
Cedar Rapids	0			2		0	0		0	1	
Davenport	0			0		0	1		0	0	
Des Moines	0		0	0	1	5	0	0	1	0	29
Sioux City	0			0		1	0		0	5	
Waterloo	0			0		2	0		0	1	
Missouri:											
Kansas City	0		0	4	4	3	0	5	0	4	85
St. Joseph											
St. Louis											
North Dakota:											
Fargo	0		0	0	0	0	0	0	0	19	8
Grand Forks	0			0		1	0		0	5	
Minot	0		0	1	0	0	0	0	0	0	5
South Dakota:											
Aberdeen	0			0		0	0		0	4	
Sioux Falls	0		0	0	0	0	0	0	0	0	8
Nebraska:											
Omaha	0		0	1	2	0	0	0	0	3	41
Kansas:											
Lawrence	0		0	1	0	0	0	0	0	11	3
Topeka	0		0	1	1	0	0	0	0	13	16
Wichita	1		0	2	4	2	0	0	0	14	27
Delaware:											
Wilmington	0		0	0	1	0	0	1	0	3	30
Maryland:											
Baltimore	6		0	5	11	5	0	9	0	123	197
Cumberland	0		0	0	0	1	0	0	0	18	14
Frederick	0		0	0	0	0	0	0	0	0	3
Dist. of Col.:											
Washington	1		0	6	8	6	0	9	6	9	136
Virginia:											
Lynchburg	0		0	1	3	0	0	0	1	7	18
Norfolk	0		0	0	1	1	0	2	0	0	16
Richmond	0		0	15	2	5	0	1	0	0	57
Roanoke	0		0	0	1	1	0	0	0	0	27
West Virginia:											
Charleston	0		0	0	0	0	0	1	1	0	15
Huntington	0			0		0	0		0	0	
Wheeling	0		0	2	0	3	0	1	0	44	17
North Carolina:											
Gastonia	0			0		0	0		0	0	
Raleigh	0		0	0	0	0	0	1	0	1	14
Wilmington	0		0	0	0	0	0	0	0	9	14
Winston-Salem	0		0	0	0	2	0	0	0	7	6
South Carolina:											
Charleston	0	7	0	0	3	0	0	0	1	0	29
Florence	0			0		0	0		0	0	13
Greenville	0		0	0	1	0	0	0	0	0	21
Georgia:											
Atlanta	1		1	0	0	5	0	4	2	18	76
Brunswick	0		0	0	0	0	0	0	0	0	4
Savannah	0		0	0	1	0	0	1	1	0	20
Florida:											
Miami	0		0	0	0	0	0	3	1	0	31
Tampa	0	1	0	1	0	0	0	2	0	1	19
Kentucky:											
Ashland	0			0		0	0		4	7	
Covington	0		0	0	0	0	0	0	0	7	15
Lexington	0		0	0	1	0	0	1	0	10	23
Louisville	3		0	5	5	4	0	5	0	56	76
Tennessee:											
Knoxville	0		0	0	0	1	0	1	1	2	24
Memphis	0	1	0	15	0	2	0	6	2	18	87
Nashville	0		0	0	2	1	0	1	0	8	52
Alabama:											
Birmingham	0		0	2	8	0	0	5	0	3	59
Mobile	0		0	0	0	0	0	0	0	0	18
Montgomery	0			0		4	0		1	0	
Arkansas:											
Fort Smith	0			0		3	0		0	0	
Little Rock	0		0	0	2	0	0	2	0	0	5

City reports for week ended July 31, 1937—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Louisiana:											
Lake Charles	0	------	0	0	0	0	0	0	0	0	2
New Orleans	4	3	0	0	11	2	0	14	9	14	160
Shreveport	0	------	0	0	3	0	0	1	1	0	53
Oklahoma:											
Muskogee	1	------	------	0	------	0	0	------	0	0	------
Oklahoma City	0	------	0	0	2	0	0	0	3	2	43
Tulsa	0	------	------	8	------	1	0	------	1	30	------
Texas:											
Dallas	3	------	0	1	3	3	0	3	0	9	59
Fort Worth	2	------	0	0	1	0	0	1	2	5	26
Galveston	0	------	0	0	2	0	0	0	3	0	17
Houston	2	------	0	0	4	1	0	7	3	0	57
San Antonio	0	------	0	2	5	1	0	8	0	1	63
Montana:											
Billings	0	------	0	0	2	0	0	0	0	0	5
Great Falls	0	------	0	0	1	0	0	0	0	10	11
Helena	0	------	0	0	0	0	1	0	0	0	3
Missoula	0	------	0	0	1	1	0	0	0	0	11
Idaho:											
Boise	0	------	0	0	1	0	0	0	0	0	5
Colorado:											
Colorado Springs	0	------	0	1	0	0	0	2	0	1	17
Denver	5	------	0	28	6	4	0	1	1	24	75
Pueblo	0	------	0	0	1	1	0	0	1	3	6
New Mexico:											
Albuquerque	0	------	0	0	1	1	0	3	0	2	17
Utah:											
Salt Lake City	0	------	0	20	0	2	0	0	0	10	26
Washington:											
Seattle	0	------	0	4	5	1	0	3	1	32	72
Spokane	0	------	0	13	2	2	0	0	0	9	30
Tacoma	0	------	0	0	2	0	0	1	1	8	26
Oregon:											
Portland	0	------	0	1	1	3	4	3	0	1	78
Salem	0	------	------	0	------	0	0	------	0	0	------
California:											
Los Angeles	4	1	0	8	8	6	4	26	2	93	319
Sacramento	1	------	0	1	0	1	0	2	0	9	21
San Francisco	0	------	0	2	2	3	0	3	0	27	140

City reports for week ended July 31, 1936—Continued

State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths	
Maine:			
Portland	0	0	1
New Hampshire:			
Nashua	0	0	1
Massachusetts:			
Boston	0	0	5
Worcester	0	0	1
Rhode Island:			
Providence	1	1	0
Connecticut:			
Hartford	0	0	1
New York:			
New York	3	0	7
Syracuse	1	0	0
New Jersey:			
Newark	0	0	2
Trenton	1	0	0
Pennsylvania:			
Philadelphia	0	0	3
Ohio:			
Cincinnati	0	0	16
Cleveland	1	0	0
Columbus	0	0	1
Indiana:			
Muncie	0	0	5
Illinois:			
Chicago	0	0	7
Michigan:			
Detroit	0	0	4
Wisconsin:			
Milwaukee	0	0	2
Minnesota:			
St. Paul	0	0	1
Missouri:			
Kansas City	0	0	3
Nebraska:			
Omaha	0	0	11
Kansas:			
Wichita	1	1	1
Maryland:			
Baltimore	2	0	6
District of Columbia:			
Washington	0	0	1
West Virginia:			
Wheeling	0	0	1
North Carolina:			
Wilmington	1	0	0
Kentucky:			
Covington	0	0	4
Louisville	0	0	3
Tennessee:			
Memphis	0	0	2
Alabama:			
Birmingham	1	0	0
Arkansas:			
Little Rock	0	0	3
Louisiana:			
New Orleans	0	0	3
Oklahoma:			
Tulsa	0	0	4
Texas:			
Dallas	0	0	3
Fort Worth	0	0	2
Houston	1	0	8
San Antonio	1	0	0
Colorado:			
Denver	1	0	0
Pueblo	0	0	2
California:			
Los Angeles	2	0	6
Sacramento	0	0	2

Encephalitis, epidemic or lethargic.—Cases: Springfield, 1; Cleveland, 1; Anderson, Ind., 1.

Pellagra.—Cases: Philadelphia, 1; Wilmington, N. C., 1; Winston-Salem, 2; Charleston, S. C., 1; Savannah, 4; Nashville, 1; Birmingham, 1; Dallas, 1; San Francisco, 1.

Rabies in man.—Death: Galveston, 1.

Typhus fever.—Cases: New York, 1; Charleston, S. C., 2; Atlanta, 2; Savannah, 3; Tampa, 1; Mobile, 1; Galveston, 1. Deaths: New York, 1.

FOREIGN AND INSULAR

BRAZIL

Santos—Poliomyelitis.—A report dated July 28, 1937, from the American Consulate in Santos, Brazil, stated that, according to unofficial information, an epidemic of poliomyelitis had appeared in Santos. More than 20 cases had been reported, with 2 deaths.

CANADA

Provinces—Communicable diseases—2 weeks ended July 17, 1937.—During the 2 weeks ended July 17, 1937, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis					2					2
Chicken pox		2		81	206	5	130	15	27	466
Diphtheria		1	4	41	24	2				72
Dysentery				1						1
Erysipelas				6	4	2		2	7	21
Influenza	1			2		7	20			30
Lethargic encephalitis					1		1			2
Measles		72	1	216	595	104	157	120	72	1,337
Mumps		9	2		98	2	26	1	22	160
Paratyphoid fever					8					8
Pneumonia	5				15		4		6	30
Poliomyelitis				2	10					12
Scarlet fever		14	11	122	115	12	24	52	18	368
Smallpox								3		3
Trachoma									2	2
Tuberculosis	10	31	42	156	128	23	2	2	29	423
Typhoid fever		1	7	17	8	1	1	1	1	37
Undulant fever				4	9	1	1			15
Whooping cough		15		417	114	95	27		11	679

CUBA

Provinces—Notifiable diseases—4 weeks ended July 24, 1937.—During the 4 weeks ended July 24, 1937, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	1	1	1	7		5	15
Chicken pox					1		1
Cerebrospinal meningitis		1					1
Diphtheria	3	15	1	3	1	5	28
Dysentery (amebic)		1					1
Dysentery (bacillary)				1	1		2
Hookworm disease	1						1
Leprosy	1	4		2	3	3	13
Malaria	65	33	25	192	70	316	701
Measles		1	13	1		1	16
Poliomyelitis	1	1				3	5
Tuberculosis	74	31	31	63	12	41	252
Typhoid fever	23	73	29	75	21	55	276
Yaws						5	5

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for July 30, 1937, pages 1054-1068. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued August 27, 1937, and thereafter, at least for the time being, in the issue published on the last Friday of each month.

Plague

Argentina.—During the second half of July 1937, 9 fatal cases of pneumonic plague were reported in the Departments of Godoy Cruz and Rivadavia, Mendoza Province, and 6 fatal cases were reported in Colonia San Juan, Figueroa Department, Santiago del Estero Province.

Hawaii Territory—Island of Hawaii—Hamakua District—Honokaa Sector.—Plague infection was reported in 1 rat on July 29, and in another rat on August 7, 1937, both from Honokaa Sector, Hamakua District, Island of Hawaii.

India—Rangoon.—On July 24, 1937, 1 case of plague was reported in Rangoon, India.

United States—Montana—Beaverhead County.—A report of plague infection in a ground squirrel in Beaverhead County, Mont., appears on page 1161 of this issue of PUBLIC HEALTH REPORTS.

United States—Nevada—Ormsby County.—A report of plague infection in fleas taken from chipmunks in Ormsby County, Nev., appears on page 1161 of this issue of PUBLIC HEALTH REPORTS.

Typhus Fever

Egypt.—During the week ended July 31, 1937, 4 cases of typhus fever with 1 death were reported in Alexandria and 1 case in Suez, Egypt. One case was reported in Ismailia during the week ended July 29.

Yellow Fever

Colombia.—Yellow fever has recently been reported in Colombia as follows: Boyaca Department, Borbur, July 12, 1937, 2 deaths; Muzo, May 28, 1 case, July 2, 1 case.

Gold Coast—Afitey.—On July 31, 1937, 1 case of yellow fever was reported in Afitey, Gold Coast.

Nigeria—Forcados.—On July 22, 1937, 1 fatal case of yellow fever was reported in Forcados, Nigeria.

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